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THE EDUCATION OF THE PROSPECTIVE MEDICAL STUDENT*

T. Lindsay Sandes, F.R.C.S.
Cape Town

Education might be defined as a knowledge of facts, and of methods and conduct, in preparation for the work of living. The major part of education was directed towards providing a living or bringing in an income. One might call this utilitarian education. On the other hand, the work of living did not cease when one shut one's shop, or office, or upon laying down the stethoscope or ophthalmoscope. One owed a duty to one's family, to the community and to the State. Education should make some preparation for these responsibilities. Owing to the length, the difficulty and the intensity of medical education, in a relative assessment of these two departments of education, an increasing want of equilibrium or balance was becoming apparent. Our medical schools were undoubtedly now, on the whole, turning out well-trained young students as doctors. This was excellent as far as it went, but it was having a serious reciprocal effect. There is a fear that the standard of general education was now becoming deficient. This deficiency will in time affect our prestige as a profession and our authority as doctors. Now consider for a moment the upbringing of a boy at school. In his early years he receives a good basic preliminary education; in his later years, certainly in many schools of the Old Country, there has been introduced of recent years a pernicious system of dividing the boys in the senior classes into two categories—the classical side and the modern side. On the classical side are collected the boys who are, for example, to become theological students intended for law, journalism, or for posts in the higher branches of the civil service. These boys study for the most part literary subjects, such as history, philosophy, the ancient classics. On the other hand, the boys destined to become chemists, engineers, geologists and doctors—their studies are mainly directed to science and mathematics. In consequence, the proper basis of education, a reasonably comprehensive foundation of knowledge, has been infringed; at an early age these children are directed towards becoming premature specialists with the narrow, the constricted, the prejudiced outlook that specializing tends to engender. The boy now passes his matriculation examination. This is a minimum standard of education for admission to the University. It is a standard which an average typist attains, a clerk in a Government office, a bank, or an insurance office, and many probationer nurses; yet it seems to be all that is required of students for the learned profession of medicine. He now enters the University but instead of mixing and communing with other undergraduates, instead of imbibing the University atmosphere, instead of getting the advantages of a broad and mixed undergraduate society of students of all types of all the professional sections and of the Arts, he at once enters the Medical School and lives in an isolated medical residence. From thence onwards he is immured in a medical monastery.

Here for the next six or seven years he perambulates the cloistered walks of the Institution. He hears nothing, he sees nothing, he inhales nothing but medical topics of one sort or another. He forgets there is a world outside. He is hardly cognizant of its existence; during those six or seven years the fleshy tablets of his mind are receiving nothing but a torrent of medical impressions. Those fleshy tablets, wax to receive and marble to retain, are permanently recording that which will affect and direct his views, his associations, and his conduct of affairs for the rest of his life. He will observe life through a polarized medium; he will see with a mental astigmatism. If he proceeds to specialize in a particular department of medicine, this imbalance of his education becomes one of a still more unstable equilibrium.

What methods can be recommended to help to correct this want of balance in his educational diet? Much as one dislikes to suggest it, after admission to the University, the prospective medical student should do one year of basic general education; in that year the subjects he studies should have no relation to, or be in any sense ancillary to, those of his medical curriculum. One recommends particularly the humanities such as history, literature, philosophy, logic and languages. I have a particular prejudice in favour of the ancient classics. This year in addition would allow a better selection of students for admission to the medical school and those who obviously had no vocation for such a professional life could more easily be separated. Furthermore, judging from 25 years' experience as a teacher of medical students, I feel less reluctant in recommending this preliminary year because those students who had a better basic education, or who were a year or two older than the average of the

* Report of the Presidential Address delivered at a meeting of the Cape Town Post-Graduate Medical Association on Thursday evening, 19 April 1951.

class, or who before had had some educational experience in other lines such as teachers, seem to me to have been better students, did better at their examinations, and made a greater success of their professional life in later years. In fact, such a year or preliminary educational years were not lost. They constituted a credit reservoir of learning and experience that was of immense advantage in the preparation for life and in practice as a doctor. I admit that this might constitute an economic hardship but I would be prepared to accept for this preliminary education a year in any employment of an educational nature which might even bring in an emolument, e.g. in place of that year, a five-months' stay in the Antarctic on a whaler or, if it could be arranged, the student should go through a course at Voortrekkerhoogte in training as a policeman, or a year in the Defence Force, or a year's experience in a foreign country where he learnt the language, or, indeed, in any employment, salaried or otherwise, provided it could be demonstrated that it was some type of educational employment, or where he enlarged his experience of men and affairs or as a citizen of the world.

The biographies of some great and successful men demonstrate how such preliminary experience not only helped but was almost a major factor in making a success of certain careers. Sir Charles James Napier, whose statue adorns Trafalgar Square, at the age of 14, was bought a commission as an ensign in the Army; his colonel, contrary to custom but being a friend of his father, sent him back home to Ireland for another four years' general education before being trained as an officer in the Army; he was severely wounded in the Peninsular wars where he lost an eye. Napier became a great general, a first-class soldier, he served with great distinction in Baluchistan, and was later a good administrator. He maintained that the success he made of his career was particularly due to the preliminary non-military education before he got submerged in his duties as a soldier. He was the only General recorded in history (since Julius Caesar) who could make a good pun in Latin.

The life of Lord Reading, the son of a Mr. Isaacs, a penurious fruit broker, is also to the point. Rufus Isaacs, finding that the living and the prospects in his father's fruit broker's office were by no means bright, went off to sea as a sailor before the mast in a windjammer. He sailed the ocean and he braved the elements as a deck-hand in this limejuicer; he became a tough, hardy, weather-beaten seaman. After many adventures the ship dropped anchor in Bombay and Rufus the young sailor got leave ashore while the ship awaited instructions. The instructions to the ship were to return home, and presently this bronzed sailor appeared in his father's office. But things were no better in the office after his years at sea; he told his father that he wanted now to study law. The old man, dismayed and surprised, explained that Rufus had neither the education, nor himself the money to permit him to study law. Rufus replied that he had made full inquiries, he had made some preparations, and he proposed to sit for the examination for the Middle Temple; although he had not much money he had sufficient saved to carry him through part of the legal training. He passed the entrance examination to the Middle Temple. He sat his lectures, he ate his dinners, and at the late age of 27 he was called to the Bar. His progress was rapid and at the age of 38, 11 years after his admission to the Bar, he took silk—almost a record. Later he entered Parliament, became a Judge, then Lord Chancellor. In due course his sovereign appointed him to the high office of Viceroy of India. Forty years after that sailor boy had climbed the seaweed covered and slippery stone steps of Bombay pier, the gateway of India, he again ascended those steps with all the emblazonry, ceremony and dignity as Viceroy of a great nation of 350 million people. Lord Reading attributed a material proportion of his success to his preliminary experiences, to the hard education he had received while a sailor on the high seas. It gave him a knowledge of men and affairs, to push aside the meretricious and to accept facts. These instances demonstrate that reasonably limited preliminary experience, as long as it was of an educational type, although it might delay the entrance to one's profession, was generally repaid with compound interest in the course of the practice of one's profession. But for the average young medical student straight from school, one year's priliminary literary education was almost a *sine qua non*.

I myself have a preference for certain subjects in this preliminary study of the prospective medical student. I would emphasize my personal regard for the value, as an educational discipline, of the ancient classics, Greek and Latin. These subjects did not seem so frequently taught in these days as they were in past generations. They were often miscalled dead languages. I would like, for a moment, to recall a little of what we owe to these ancient civilizations.

Greece, in the fifth century B.C., set standards of art, architecture, oratory, philosophy, drama, literature, logics, historical records, science and scientific research, poetry, and principles of democracy that have rarely been approached and never exceeded since those long ago times. Ancient Greece gave us the nucleus and the substance of the western civilization on which the best of modern scientific and ethical progress depends. When Greece was overwhelmed by the growing Roman Empire, that stern military administration recognized the great essential value of Grecian civilization. Rome emulated and absorbed it in its own life and institutions. Furthermore, the sons of the nobles of the Roman Empire and of those who could afford it, were sent to Greece to learn the language and to absorb the culture which the Romans deeply appreciated. So that through the Roman Empire we have received and impounded a great deal of the culture and the civilization that was Grecian and on which the West is founded. The Roman Empire lasted 1,000 years. It extended from the lowlands of Scotland in the west to the Tigris and Euphrates in the east; from the Danube, the Rhine and the Baltic in the north. It embraced the North African littoral to the burning deserts of the south. The engineering of Rome, its buildings, bridges, its roads and highways, are there to be admired to this day. Roads that travel almost in straight lines from the capital to the remotest marches of the Empire; over mountains, across valleys, by bridges and viaducts. Communication was swift and efficient. Roman law first enscrolled on the 12 tables in the early days of the Roman kings and later enlarged and amended by the Republic. Ultimately Justinian in A.D. 533, after 1,300 years of legislation,

promulgated an imperial statute comprehending a simplified code and digest of valid laws. Those laws form the basis of the legal systems of all Southern Europe, the British Isles and of all the Americas. Again in the fortified outposts of the Empire the Roman soldiers were good ambassadors and agents. The people and the peasantry, whether in Spain or Portugal or Gaul or Belgium or Italy, learnt to speak the soldiers' and traders' Latin. It was not the classical Latin of Cicero's orations, nor the sonorous phrases of Virgil's hexameters. It was the common speech of the ordinary people. In each of these countries this speech was gradually adopted, gradually spread and adapted, affected and modified by local intonation and dialect and developed into the languages of each of these nations. Half the words of English which I myself am now using were derived directly or indirectly from Latin; and the civilized speech of all the Americas, from Canada to Magellan, was either a modified Latin through the Spanish or Portuguese, or in the English-speaking parts, half is derived from Latin. The civilization of ancient Rome in which the Grecian standards were absorbed was at a very high level in the best days of the Empire. It was said that no Roman citizen was illiterate. He could read and write and figure. He had a definite standard of education; the only members of the Roman Empire who did not rise to those standards were the lowest class of slaves. The better class of slaves were often highly educated and acted as teachers and tutors. But the lowest class of slaves, e.g. coolies and labourers, did not have this standard nor did the recently conquered barbarians. The very word barbarian is imitative of the rude speech of a wild, illiterate race. It was a highly prized and privileged honour, but not without its obligations, to be able to say *Civis Romanus sum*.

About the fifth century A.D. the furious and repeated inroads of the vandals, the Goths and the Huns commenced to press back the Roman Empire, already enfeebled from many intrinsic ailments. Western civilization commenced to collapse. The Dark Ages had begun. For four or five centuries it looked as if civilization would be entirely submerged and destroyed; the world, indeed, nearly slipped back to prehomeric standards. But all through those dark ages civilization nearly asphyxiated, buried and hidden, still maintained a spark of life; credit for the nurture and maintenance of that spark of life must be given in the first place to the monasteries—the monasteries of the great Catholic brotherhoods. Their records were kept in classical Latin, their communications and commerce were conducted in that speech and while the Latin language survived it could always bring back the immense documentary records and literature of the lost civilization. About the tenth to the twelfth centuries many of these monasteries began to develop into educational institutions and these were the beginnings of Universities. Padua, Salamanca, Prague, Heidelberg, Paris, Vienna, Oxford, Cambridge, St. Andrews—Salerno and Bologna Universities are said to have the distinction of being the first. The renaissance had dawned. Since then our civilization has developed rapidly. It is founded upon the principles, the precepts, the methods, and the culture derived in the first place from ancient Greece and Rome. The very language of scientists, of Newton, of Harvey, of Gillileo, up to mid-Victorian times, was classical Latin.

I must refer to the pleasure and refreshment that I derive from the well-thumbed textbooks of the classics which I still have from my far-off youth. I admit I am prejudiced in favour of the classics. I am convinced of their basic educational value. Though not so readily convertible into commercial barter as technical scientific attainments, they had an incontrovertible claim to be considered in any educational curriculum. It was for these reasons that I recommend them in any preliminary course before students enter their medical school in an attempt to balance the overweighted scientific studies which the medical student must later undertake.

Such a pre-medical educational year should be founded on the humanities. What shall it profit a man if he gain the whole world and lose his own soul.

NEW PREPARATIONS AND APPLIANCES

TENAMID: A NEW ANTHELMINTIC

Tenamid, the recently discovered anthelmintic preparation of the Schering Corporation, allows for the rapid and effective oral therapy of tenia infestation of the intestines.

Chemically known as β-phenyl-δ-carboxy- (3, 5-diiodo-4-hydroxy phenyl) ethane, *Tenamid* has been proved effective against *Taenia solium*, *Taenia nana*, *Taenia saginata*, *Necator americanus*, *Hymenolepis*, *Dipylidium caninum*, *Bothriocephalus latus*, *Trichocephalus* and *Ascaris*, in a high percentage of cases reported.

Usually one course of treatment is sufficient to expel the parasites completely. A second or even a third course of treatment may be instituted at a 6- to 8-day interval, in that rare case which does not respond to a single course.

Tenamid is in tablet form and administration is simple. No special diet or purgatives are necessary before treatment. The successful therapeutic results obtained are marked by a complete absence of detrimental secondary effects.

Tenamid tablets each 0.5 gram are available in tubes of 12 and bottles of 100.

Manufactured in the Union of South Africa by Scherag (Pty.) Limited, Johannesburg, for and under the formula and technical supervision of Schering Corporation, Bloomfield, N.J.

ABSTRACTS

Anti-Pernicious Anaemia Factor and White-cell Count. J. Dedichen and P. Laland (1949): Lancet, **257**, 282.

As already found with folic acid, pure crystalline Vitamin B_{12} (40 micrograms) had no effect within one day on the leucocyte-count in one normal patient. Following a subsequent dose of 10 cc. liver extract, leucocytosis occurred promptly.

Vitamin B_{12}. D. M. Dunlop and W. M. Wilson (1949): Lancet, **256**, 754.

An extract from a strain of Streptomyces after assaying microbiologically for Vitamin B_{12} activity was used to treat two cases of Addisonian pernicious anaemia. The response was equivalent to that expected from a potent liver extract.

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VAN DIE REDAKSIE

ONHOORBARE KLANKGOLWE EN BEHANDELING

Die verwarmende invloed van onhoorbare klankgolwe is biologies gesproke van weinig belang. Van groter belang egter is die eienskap wat dit besit om 'n vermeerderde bloedrykheid te veroorsaak, terwyl daar ook aanspraak op gemaak word dat hierdie golwe 'n byna spesifiek meganiese invloed besit op die vog in die weefselruimtes, die verspreiding daarvan, met deurgang deur weefselvliese, asook op die gemiese toestande en dié van die struktuur van die sel. Op grond van hierdie eienskappe is daar al aanspraak op gemaak dat gunstige geneeskundige uitslae behaal is by die behandeling van gewrigs-, senuwee-, en veselweefselstoestande. Dit het in die berigte nie ontbreek aan verslae nie, omtrent die goeie subjektiewe gevolge by gevalle van „ulcus duodeni, vergroeiings in die die buikholte, galblaasontsteking . . . en senuweepyn van die buikwand'.[1] Die invloed van behandeling met onhoorbare klankgolwe is al toegeskryf geword aan mikromassering van die selle sowel as aan verhoging in die temperatuur van die weefsels sowel as aan kolloidaalveranderinge.

Terwyl daar weinig twyfel kan bestaan dat, onder proefondervindelike omstandighede, hierdie golwe 'n mees diepgaande invloed uitoefen op die weefsels, is daar 'n neiging by die meerderheid van hierdie verslae, waarin daar aanspraak gemaak word op gunstige geneeskundige resultate, om van uit Westelike Europa te kom. In Engelse en Amerikaanse hande was die uitslae nie van bepaalde aard of enig nie en het dus nie aangetoon of hierdie vorm van behandeling (wat geensins sonder gevaar is nie) bepaalde vooruitgang van enige soort verteenwoordig, bo enige van die bestaande wyses van behandeling nie.

DIE GEVARE

Die onhoorbare golwe mag, wanneer dit gerig word op die plexus brachialis, wederkerende hartkloppings en hartkramp veroorsaak. Alhoewel daar al aanspraak op gemaak is dat goeie uitslae verkry word by die behandeling van sere aan die been, bly daar nóg die gedurige gevaar dat plaaslike bloedklonte in beweging gebring kan word. Die golwe kan ook, as gevolg van hul invloed op die geslagskliere, onvrugbaarheid veroorsaak; hulle kan die ontwikkeling van die groeiende gebeente by kinders hinder en maak dit vir die werker iets gewaagds. Dit is dáárom duidelik dat die werktuie vir die vervaardiging van hierdie golwe as uiters gevaarlik beskou moet word. Daar kan geen verskoning voor wees dat leke toegelaat sal word om hierdie dinge te hanteer nie en dit is wenslik dat selfs lede van die mediese beroep die toestel alleenlik onder lisensie sal gebruik.

1. Excerpta Medica (1951) Afdeling VI (Internal Medicine), **5**, 317.

EDITORIAL

ULTRASONIC WAVES AND TREATMENT

The thermogenic effect of ultrasonic waves is biologically not of much significance. More important is the property of inducing hyperaemia and it has also been claimed that these waves have almost a specific mechanical effect upon the circulation of fluid in the tissue spaces, diffusion through tissue membranes and the chemical and structural conditions in the cells. It is on the basis of these properties that favourable therapeutic results have been claimed in the treatment of arthritic, neuritic and fibrositic conditions. Reports have not been lacking about good subjective results in cases of 'duodenal ulcer, abdominal adhesions, cholecystitis . . . and neuralgia of the abdominal wall'.[1] The effect of ultrasonic therapy has been ascribed to micromassage of the cells as well as an increase in the temperature of the tissues and to colloidal changes.

While there can be little doubt that under experimental conditions these waves exercise a most profound effect on tissues, most of the reports claiming favourable therapeutic results tend to come from Western Europe. In English and American hands the results have not been specific or unique and have not indicated that this form of treatment (which is not without its dangers) represents any advance of any kind on any of the existing methods in current use.

DANGERS

The ultrasonic waves may, when directed to the brachial plexus, produce paroxysmal tachycardia and angina pectoris. Although it has been claimed to have good results in the treatment of ulcers of the leg, there is the ever-present danger of mobilizing regional thrombi. The waves can also, by their action on the gonads, produce sterility; they can interfere with the development of growing bones in children and place the operator of the instrument at risk. It is clear, therefore, that instruments for producing these waves must be regarded as highly dangerous. There can be no excuse for permitting their use by lay persons and it is desirable that even members of the medical profession should use the apparatus only under licence.

1. Excerpta Medica, 1951, Section VI (Internal Medicine), **5**, 317.

Terwyl dit twyfelbaar lyk of daar enige geneeskundige deug bestaan vir die aanwendig van onhoorbare golwe en omdat daar nog geen vaste wetenskaplike saak voor uitgemaak is vir toeskrywing aan hierdie golwe van enige geneeskundige voortreflikheid op spesifiekheid nie, behoort ons uiters versigtig te wees t.o.v. die binnelating van hierdie gevaarlike instrumente na die Unie en, veral, die hantering daarvan deur leke. Van welke aard die aansprake ook al sal mag wees, moet hul op hierdie stadium gewend word na die gebied van die navorsing liewers as na dié van die kliniese praktyk.

As it seems doubtful whether any special therapeutic virtue is inherent in the use of ultrasonic waves and because no sound scientific case has yet been made out for giving these waves any therapeutic pre-eminence or specificity, we should be most circumspect about the introduction of these dangerous instruments into the Union and, in particular, their use by inexpert lay persons. Whatever claims are to be made should at this stage still be directed to the field of research rather than clinical practice.

BILHARZIASIS IN THE TRANSVAAL

S. ANNECKE, M.A. (CAPE TOWN), M.B., B.S. (DURHAM), B.Hy. (DURHAM), D.P.H. (LOND.), D.T.M. & H. (LOND.) M.R.C.S., L.R.C.P.

and

P. N. B. PEACOCK, M.B., CH.B. (CAPE TOWN), D.P.H. (CAPE TOWN)

Union Health Department, Tzaneen, Transvaal

(*Concluded from page* 680)

(c) *Distribution of Bilharziasis* (S. Haematobium *only*). In 1937-1938 a somewhat limited survey to determine the infectivity rate amongst Native school-children was undertaken. The results of this survey appears in Table E.

A much more extensive survey which covered nearly the whole of Transvaal where bilharziasis occurs, followed in 1948. Whereas the 1937-1938 survey dealt primarily with Natives the later one included numbers of European school-children as well.

Attached Tables E and F reflect the distribution of bilharziasis amongst Native school-children in the Transvaal in the years 1937-1938 and 1948-1950 respectively. Table G conveys the same information for European school-children in the years 1948-1950. To simplify these tables a number of schools from each district have been lumped together with the result that the very high percentage incidence (90% +) found in certain individual schools is not shown. (In the 1937-1939 figures, when smaller numbers were examined the higher percentages are more obvious.) The attached map shows those parts of each area where the percentage incidence was above or below the pooled figures given in the latter. Our criterion for considering a subject as having Bilharzia was the microscopical identification of viable Schistosome ova in a single centrifuged terminal urine specimen collected under supervision after moderate exercise as recommended by Bennie.[17]

We notice from the tables that:

i. Bilharzia is widely distributed amongst Native school-children over the Northern and Eastern Transvaal, with infectivity rates of up to 80%.

ii. The surveys of 1948–1950 correspond closely with the 1937–1938 surveys.

iii. The highest incidence of bilharziasis exists in the North and North-Eastern Transvaal Lowveld and portion of the Western areas of the North Transvaal with less in the intervening Highveld (Map, Fig. 1). This map also shows how patchy the distribution of bilharziasis is in the Northern Transvaal.

iv. The incidence of Bilharzia amongst European school-children is much lower than amongst Native school-children, but the occurrence of the disease is as widespread.

It should be noted that those figures only refer to a selected group (i.e. children) and they cannot be considered as giving the general incidence amongst the whole population.

The accompanying map, referred to above, deals only with the distribution of *S. Haematobium* in the Transvaal and shows many areas where more than 60% of the Native school-children are infected (sometimes 60% and over). The distribution of *S. Mansoni* has not been explored to any great extent but it is known to occur fairly frequently with the majority of the known cases concentrated in the Transvaal Lowveld to the east of the Drakensberg Mountains.

TABLE E: URINE EXAMINATIONS NATIVE SCHOOL-GOING CHILDREN 1937-1938

Zone	*Locality*	*Number Examined*	*% Positive*
Zone I	Shulivane	322	67
	Sibasa	1,577	31
Zone IV	Nelspruit	119	75
	Nelspruit Location	144	88
	White River	391	50
	Nelspruit Town	1,016	30
	Karino	460	49
	Bosbokrand	426	53

TABLE F: URINE EXAMINATIONS (NATIVE SCHOOL-GOING CHILDREN)

Zone	Magisterial District	Number Examined	Percentage Positive
Zone I	*Letaba:*		
	Klein Letaba 6 schools	321	35
	Moketsi 2 schools	192	24
	1 school	100	23
	Letaba 10 schools	876	50
	Olifants 4 schools	273	53
Zone III	*Waterberg:*		
	Sanddrift	47	51
	Roodepoort	101	27
	Middelfontein	101	50
	Rustenburg:		
	Marikana, Tweedepoort and other schools	1,178	33
	Groblersdal:		
	Dennilton	283	32
	Ottensville	109	30
Zone II	*Pietersburg:*		
	Olifants 2 schools	306	25
	Cheunies 2 schools	230	6
	Marabastad 2 schools	193	4
	Pietersburg 5 schools	520	4
	North West 12 schools	922	7
	Potgietersrust:		
	7 schools	813	13
	Lydenburg:		
	(Highveld south of Steelpoort River) 3 schools	218	19
	Lowveld, north of Steelpoort River	777	13
Zone IV	*Eastern Transvaal:*		
	Lowveld 11 schools	700	56

TABLE G: URINE EXAMINATIONS: EUROPEAN SCHOOL-CHILDREN

Zone	Magisterial District	Number Examined	% Positive	Zone	Magisterial District	Number Examined	% Positive
I	*Letaba:*			II	*Lydenburg:*		
	Merensky School Farm	295	10		Origstad	130	17·4
					Rustplaas	32	3·1
					Three other schools	183	5·2
III	*Groblersdal* and surrounding areas:				De Groot Boom		
					Krugerspoort		
					Burgersfort		
	Dennilton, Marble Hall, Groblersdal, Waterberg, Nine schools	668	11·2	IV	*Eastern Transvaal:*		
					Two schools (Komatipoort, Elandshoek)	200	5·2
	Rustenburg (Schools 4)	638	5		Barberton	558	5·7
	Paul Kruger, Rustenburg, Kafferskraal, Elandskraal	1,563	4·4		Nelspruit	703	7·1

It is of marked practical importance to note that the distribution of bilharziasis is considerably more widespread than the distribution of the Bilharzia snail which is, after all, limited to the water sources or water holdings (dams, etc.) which are often a long distance apart. This means that in some areas, especially in the drier country to the west of the Drakensberge, the clearance of a relatively small number of streams and dams will prevent the transmission of bilharziasis over a large block of country.

DISCUSSION

Numerous problems and obstacles have been encountered. Most of these seem surmountable. Others will still have to be dealt with. It would be appropriate to cite just a few of these obstacles and it will readily be appreciated that Bilharzia control is by no means as straightforward undertaking as was malaria control.

1. The success of any scheme for Bilharzia control must largely depend upon the willing co-operation of the farming community. In the Northern and North-Eastern Transvaal it has taken us many years to convince these farmers—individually, and through their various associations—that our mosquito larvicides are not harmful to their stock and their crops on which most of them are solely dependant for their existence. Sulphation offers the same problem and we expect the same or more difficulty in persuading the farmer that copper sulphate will have no deleterious effects. Fortunately we can give this assurance with some confidence as our biological and chemical tests

have shown (see back in this paper) how rapidly copper sulphate is rendered innocuous when added to natural collections of water.

2. During the rainy months (November to March) large numbers of snails are found in the grass pans which are such a distinctive feature of the Northern Transvaal. These snails are mostly *Bulinus tropicus*, but from 10% to 20% (see Table D) are the proven Bilharzia vector, *Physopsis africana*. Most of these pans dry up a few weeks to a few months after the last summer rains and may remain dry for many months before they are refilled with water. As soon as this happens the snail population re-appears. Where do these snails come from? We have found that these snails, with their openings sealed with epiphragms made largely of mud, aestivate in their thousands hidden in the depths of the dry tufts of grass and in between the grass roots. When placed in water the artificial epiphragm dissolves and normal life is resumed. We have raised new colonies of snails (*Bulinus tropicus*) from specimens taken from these pans as long as 18 months after the last water has dried up. Shousha similarly records that snails in Egypt and the Sudan will survive drying for 12 months, though apparently the mechanism is somewhat different. We have not yet found Planorbis to aestivate in this way, though it is possible that they do.

The resistance to drying naturally makes control more difficult as the aestivating snail is highly inaccessible to us and our molluscicides. It suggests that the best time for molluscicidal work in this type of water collection is when the water is at its highest as living snails do not appear to bury themselves when water is available.

3. Of at least as great significance as the incidence of Bilharzia amongst the human and cattle population is the incidence of bilharziasis amongst the natural snail population. This we have been unable to establish, as we cannot under field conditions differentiate between the various cercariae found, except to say that they are bifurcus or afurcus. De Meillon[7] has previously commented on the difficulties found in identifying cercariae found under natural conditions on morphological grounds. It is considered though that for practical purposes, the recovery of typical forked-tail cercariae (either by placing the snail in fresh water—the ideal temperature is some 80° F though we have found shedding at as low as 50° F or by dissecting the snail) from known Bilharzia vectors (*Physopsis africana* and *Planorbis pfeifferi*) in an area where human bilharziasis occurs, is sufficient presumptive evidence to justify our considering these snails as dangerous and worth control. Any field work in this direction will, however, be of limited value until such time as all cercariae can be accurately identified.

It was a matter of regret to us that DDT—which we used extensively as a mosquito larvicide—is not sufficiently cercaricidal to be of value in the control of bilharziasis—as shown by Kuntz and Stirwalt.[18] Incidentally, Jones and Brady[19] showed that copper sulphate was only an effective cercarcide in concentrations above 50 p.p.m. and McMullin and Ingals[20] failed to kill the cercariae of *B. japonica* with concentration of copper arsenate, copper cyanamide or Paris green of up to 100 p.p.m. Gonzalez, Biaggi and Leon[21] have, however, shown that a chlorine residual of 0.5 p.p.m. for 20 minutes will kill *B. mansoni* cercariae and this is of practical importance.

As yet the Department of Health has undertaken no systematic campaign to control bilharziasis and the work reported in this paper was conducted concurrently with the multitudinous other duties, especially malaria control, which keep the Malaria Unit of the Department busy in this part of the Union. It is hoped, nonetheless, that it will soon be possible to undertake a real control programme and in the meanwhile our preliminary work is continuing along the lines which appear to have the most immediate practical significance and in close liaison with biologists and all those interested in the preservation of our fauna.

It must be emphasized that the work was largely voluntary in nature and it was only the willing and enthusiastic team-work of all members of our unit—field staff, technical and administrative—which made it possible. To them we extend our sincere appreciation.

SUMMARY

1. Bilharziasis is known to occur in varying degrees of severity in the Northern and Eastern Transvaal—an area of over 60,000 square miles. The reasons are discussed why molluscicidal work is the only possible practical method by which the disease can be controlled in this area.

2. The distribution of the more important fresh-water snails is given for different parts of the Transvaal for the four quarters of 1950, and the distribution in various habitats is given for the known vectors *Physopsis africana* and *Planorbis pfeifferi* as they occurred during these four quarters. The close relationship between the occurrence of *Physopsis africana* and the incidence of *B. haematobium* is demonstrated and discussed.

3. Various molluscicides are discussed, and the opinion is given that copper sulphate is at present the molluscicide of choice (within economic limits). The instability of copper sulphate is demonstrated, and it is suggested how this chemical may best be used under our conditions.

4. Urine examinations done during the years 1937-1938 and 1948-1950 give percentage incidences of bilharziasis among Native school-children which vary from 88% to 4%, with the highest incidence in the low-lying country to the east of the Drakensberg mountains. The corresponding figures for European school-children vary from 17% to 3%. The patchy distribution is emphasized.

5. Problems created by the farming community's fear that copper sulphate may injure their stock or their crops is discussed.

6. The aestivation habits of certain snails are described, and a mention is made of the difficulty of identifying cercariae obtained from snails collected in their natural habitat.

7. It is indicated that this work has been of a preliminary and largely voluntary nature.

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ASPECTS OF CRANIO-CEREBRAL TRAUMA

I. THE SURGICAL ANATOMY AND PHYSIOLOGY OF CRANIO-CEREBRAL TRAUMA *

DAVID A. MUSKAT, CH.M.
General Hospital, Johannesburg

SURGICAL ANATOMY

A proper conception of the mechanics and pathological sequences of cranio-cerebral injuries requires a knowledge of certain intracranial features. Trauma, especially through its widespread effects, can disorganize any and every part of the cranial contents, so that the anatomical configuration not only has an important bearing on the pathology of head injuries but may, and often does have a direct bearing on the death of the patient. Certain aspects of cranial anatomy therefore merit discussion.

The Skull. Except for certain thinned-out areas at the base, the inner and outer surfaces are condensed to form compact expansions of bone (the inner and outer tables). The outer table is thicker and roughened to allow for muscular and fascial attachments; the inner table is harder, approaching ivoriness in parts, yet, for this very fact, possessed of a certain brittleness in structure. The diploeic veins, in the soft, gritty and porous diploe between the tables, are wide, thin-walled and easily torn. Owing to adhesion to the surrounding diploe, they are unable to retract and so bleed profusely when torn. They lack valves and at intervals communicate with surface veins via the emissary veins, and with the underlying meningeal veins.

The bones of the vault are developed in membrane and retain a certain amount of elasticity; but the bones of the base, developed in cartilage, lack this feature, being more brittle. At places the bones of the base are paper-thin and may even be absent and replaced by a membranous layer of fibrous tissue (e.g. the cribriform plate).

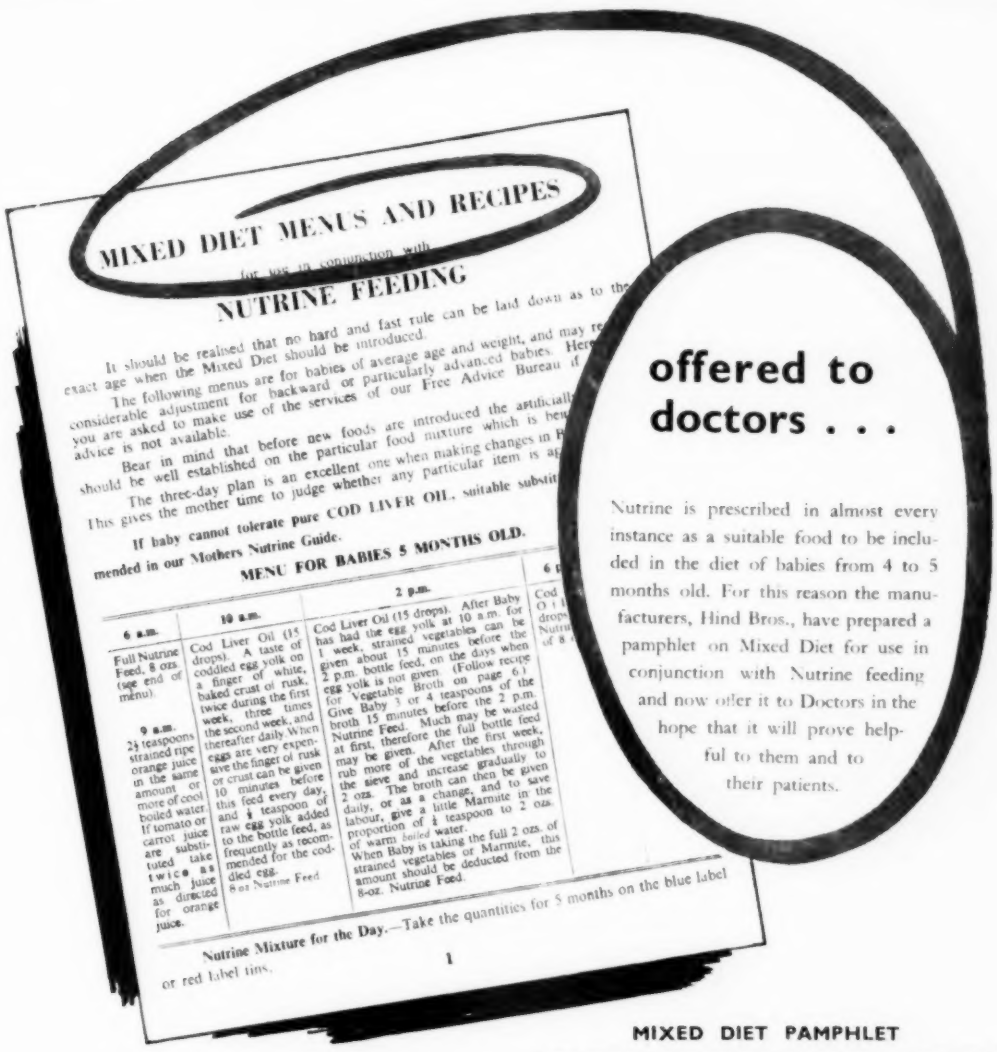
The diploe has a certain amount of protective function, acting as a medium for the dissipation of forces applied to the exterior (*cf.* shatterproof glass—Dandy, 1947). However, more important protection is rendered by longitudinal and transverse thickenings of bone, giving it a buttress-like support. These occur at the glabella, the external angular process, the mastoid bones, the external occipital protuberance, the supraorbital ridges, the temporal crests, the occipital line, the longitudinal arch, the thick posterior rim of the foramen magnum, the petrosal ridges and, more anteriorly, the sphenoid bones. Although these buttresses are thickened parts of the bony structure, they exhibit a tendency to brittleness, accounting for the frequency with which fracture-lines cross them. It is more usual, however, for fissures to be deflected and deviated to points of weakness such as foramina, the cribriform plates, the pituitary fossa, the lateral parts of the petrous bone, the sphenoidal fissure, the roofs of the orbits and the tegmen tympani (Rowbotham, 1949).

In the infant the vault of the skull is large compared to the base and the membranous layer between the sutures is of considerable thickness. The mobility of the bony plates and the fibrous attachments of the vault tend to confine the effect of violence to the bones primarily struck, with but little transmission of the force to other parts of the cranium (Vance, 1927). This very fact, however, renders the underlying soft brain more vulnerable to injury. With increasing age sutural lines are lost, the skull bones become more brittle and thicker, mainly due to deposition of bone on the inner table to replace the shrinkage of the brain.

It is generally taught that the Bantu skull demonstrates differences in texture and thickness from that of the average European. Certain craniological differences undoubtedly occur. However, a small sphere exhibits greater solidarity than a large one, and some of the alleged increased resistance of the Bantu skull to force, may be due to the fact that the average weight of the European brain is 1,475 gm. compared to that of 1,331 gm. in the Negro (Weaver, 1930). Personal experience in operating on the Bantu skull, observation of skulls at autopsy, perusal of autopsy records and the observation of experienced pathologists, have failed to justify the conviction that the skull of the average Bantu is thicker or tougher than that of the average European.

The Dura Mater. This thick, resistant, inelastic membrane forms a lining for the bony cavity and a mechanical support for the brain and the large venous sinuses. It has the character and functions of a fibroblastic membrane (Leary and Edwards, 1933). The outer layer is firmly adherent to the skull, particularly in the very young (*cf.*

* The References will be published at the end of the concluding article in this series.



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the rarity of extradural haematoma at this age); adhesion diminishes with age but in the very old it again tends to be more marked. Through the various adhesions pass minute meningeal arterial twigs and fibrous processes which tear in the process of separation of the dura from the inner table.

Of the various dural septa the *tentorium cerebelli* assumes important significance in cranio-cerebral pathology. The concave free edge, anteriorly, forms the margin of a space—the incisura tentorii—in which lie the midbrain, the basilar vessels, the posterior cerebral vessels, the oculomotor nerves and the anterior part of the superior vermis of the cerebellum. The space of Bichat, between the mid-brain and the sharp crescentic border, is minimal, allowing for the free flow of cerebrospinal fluid between the infratentorial and the supratentorial cisterns. A firm, resistant structure thus effectively divides two important regions of the brain from each other, viz. the infratentorial compartment containing the 'vital structures' (medulla, pons, cerebellum) from the supratentorial compartment with its contained cerebral hemispheres, a relatively narrow, soft core of brain tissue intervening between the two.

The dura mater is richly supplied with arteries and veins, particularly in the fossae. Anteriorly it is supplied by meningeal branches of the ethmoidal and internal carotid arteries. In the middle cranial fossa are the middle and accessory meningeal branches of the internal maxillary artery, and branches of the ascending pharyngeal and internal carotid arteries. More posteriorly we find branches of the occipital, and anterior and posterior meningeal branches of the vertebral arteries. The great vascularity of this dense, adherent membrane is one of the major contributing reasons for the gradually or rapidly increasing space-occupying lesions created by haemorrhage, the ever-increasing haematoma breaking down more and more communicating twigs, giving rise to what one might appropriately call a vicious expansion. The dura is particularly adherent in the region of the cribriform plate, where it is also thinnest. It is, moreover, very firmly anchored by its extensions through the bone along the olfactory nerves (Cairns, 1937).

The *subdural space* can be likened to the peritoneal and pleural spaces, where the smooth surfaces are separated by a thin film of moisture to allow for movements of organs; it must be remembered that the brain is a constantly moving (i.e. pulsatile) organ. The space is potential, being in life merely a cleft. Embryologically the formation of the space probably derives from the separation of the ectodermal pia-arachnoid from the mesenchymal inner layer of the dura mater: hence the difference in properties of these membranes. The pia-arachnoid is poorly vascularized and has weak fibroblastic properties, whereas the dura reacts strongly to infection and trauma and can organize and remove blood from the subdural space (Leary and Edwards, 1933).

Certain Important Vessels. The anterior branch of the middle meningeal artery crosses the great wing of the sphenoid and in its anterior inferior angle, *with its venae comites*, grooves the bone deeply or may even be completely enclosed in a bony tunnel for a part of its course. It and its main branch follow roughly the line of the precentral sulcus. The artery is closely adherent to the dura, particularly where it is semi- or completely encased by bone (in 60% of cases—Gurdjian and Webster, 1942) so that it is almost impossible to separate the dura from the skull without causing rupture of the artery or its companionate veins, the thin-walled nature of the latter facilitating injury, with resultant extradural or even subdural haemorrhage.

The *posterior cerebral arteries* are the terminal branches of the basilar artery. Each artery passes lateral to and immediately above the oculomotor nerve, where it lies in very close relation to the free edge of the tentorium cerebelli. Winding round the cerebral peduncle it reaches the under surface of the cerebrum to supply the temporal and occipital lobes. From its position between the mid-brain and the third nerve medially and the free edge of the tentorium laterally, it can readily be understood how a supratentorial herniation of the uncus can squeeze and even occlude the artery with resultant ischaemia or infarction of the area of brain supplied by it (including the cortical visual areas).

The Cortical Veins. Of surgical importance are the superior cerebral veins draining the supero-lateral and medial surfaces of each hemisphere. The anterior veins drain into the longitudinal sinus almost at right angles, whilst the larger posterior ones run obliquely forwards, upwards and medially, thus entering the sinus in a direction opposite to the flow of blood inside it. The relatively unsupported veins are thin walled and elastic, permitting participation in the normal variations of intracranial pressure. They leave their partially protected beds in the sulci to enter the sinuses by piercing the arachnoid mater and traversing the subdural space for a short but variable distance, finally to enter the longitudinal sinus, a relatively fixed point. The transition between a potentially moving cortical surface to a fixed dural point is an important aetiological factor in the causation of subdural haematomata.

The *uncus* is an important portion of brain tissue, at times playing a major role in abnormal intracranial hydrodynamics. It is the anterior recurved end of the hippocampal gyrus, lying in juxtaposition to the third nerve and in close relation to the anterior end of the tentorium cerebelli, and abutting on the side of the mid-brain. In conditions of increased intracranial pressure above the tentorium, the uncus, from its position, is forced downwards into the space of Bichat, nipping the third nerve between the midbrain and tentorium, and is responsible for certain important clinico-pathological consequences.

SURGICAL PHYSIOLOGY

1. *The Cerebrospinal Fluid.* The inferior surfaces of the frontal and temporal lobes (and the tips of their poles) are separated from the floor of the skull by only a thin layer of intervening fluid. Absence of cerebrospinal fluid cushioning is one of the factors predisposing these regions to the direct and indirect effects of trauma. Although it is generally accepted that absorption of fluid is effected by the arachnoidal villi and granulations, there are some who believe that there is a direct passage of cerebrospinal fluid into the blood stream (Howe, 1928; Dandy, 1947), probably by osmosis. A major function of the cerebrospinal fluid is its cushioning or dampening effect in

protecting the brain from external jars and forces. In order to maintain this function a certain amount of pressure is required.

2. *The Intracranial Pressure.* It has long been taught (Monroe-Kellie doctrine) that the total volume of the cranial contents is fixed by the rigid confines of the bony walls. In order to maintain pressure equilibrium, a change in any one component should be associated with a reciprocal change in some other, i.e. brain, cerebrospinal fluid or blood. In contrast to this older conception of the closed box, the cranio-vertebral cavity is now recognized to have a certain amount of elasticity, due particularly to the remarkable resiliency of the cerebral circulation (Loman, 1938). One can regard the cranio-vertebral contents as comprising two types of elements.

1. Static or inelastic;

2. Dynamic or elastic (mainly the vessels, particularly the thin-walled veins).

The normal limits of the cerebrospinal fluid pressure range from 50-200 mm. water, i.e. with the patient in the lateral horizontal position. In the erect or non-horizontal position the pressure is not so much related to the column of cerebrospinal fluid above the needle as to the hydrostatic and elastic changes occurring in the venous wall surrounding the fluid, e.g. upward tilting causes a lowering of the venous and hence of the cerebrospinal fluid pressure (Loman). In the erect position the normal adult intracranial pressure varies from 50-150 mm. water below atmospheric pressure. The zero point is about the level of the lower cervical vertebrae (Munro, 1938).

3. *The Role of Vascular Factors.* Opinion favours a sympathetic nervous control of the cerebral blood vessels. In addition, contraction of the smaller vessels can occur independently of the nervous stimulation, as, e.g. proved by Echlin (1942) who showed that pial vessels can contract vigorously locally when stimulated under experimental conditions. The cerebrospinal fluid pressure is closely related to the venous pressure and the cerebral blood flow to the arterial pressure, the combination of these two factors tending to keep oxygenation of the brain at a uniform level. Paradoxically, when the systemic blood pressure falls there occurs a marked vasodilatation, the critical level at which this occurs being about 70-80 mm. Hg (Howe, 1928). The vasodilatation in the face of a falling blood pressure can actually provide a greater blood flow, provided the fall is not too great, e.g. by histamine or amyl-nitrate (Loman); CO_2 is a potent vasodilator, and alcohol to a lesser extent, but as a result of the concomitant general vasodilatation, the blood pressure may be sufficiently lowered to cause a decrease in the cerebral blood flow. Vasodilatation can also occur reflexly in concussion by reflex stimulation of the vagus nerves (Denny, Brown and Russel, 1941), probably via the parasympathetic fibres of the seventh cranial nerves (Forbes, 1940). Scheinker (1944) considers vasoparalysis a frequent reaction of the blood vessels of the nervous system, caused by acute trauma or intoxication. This circulatory stasis or vascular damage, once established, would result in progressive cerebral ischaemia, anoxia and oedema (Masserman, 1934).

Oedema tends to reduce the cerebral blood flow by compression-narrowing of the capillaries and venules. Loss of fluid from the perivascular spaces, on the other hand, tends to dilate the capillaries and veins but, though increasing the amount of blood in the vessels, will not necessarily increase the flow, particularly when, as in these cases there is often a generalized loss of fluid, causing not only a lowering of systemic blood pressure but also an increased viscosity and resultant peripheral resistance (Forbes, 1940). Cerebral blood flow is also reduced by a rise in the general intracranial pressure above 300 mm. water (Wright, 1945). Adequate blood flow is important as the brain has a high level of metabolism, causing venous blood leaving the brain to be extensively reduced. Energy utilization in the brain is entirely provided by carbohydrates and it is here that the role of vitamins, particularly the B group, is so important. These factors influence O_2 consumption greatly and therefore are intimately related to the blood flow.

Any condition causing compression of the cerebral veins or impeding the venous outflow from the brain will cause a rise in intracranial pressure. This occurs, e.g. in the well-known Queckenstedt test, also in the head-down position and conditions increasing the intrathoracic pressure, e.g. coughing, straining at stool, laughing, etc. A generalized rise in intracranial pressure results in the following sequence of events (Forbes, 1940): The compression of the veins results in a slowing of the rate of blood flow and a decrease in the volume of output. Venous stasis follows and capillary pressure rises sufficiently to overcome the obstruction and the circulation is resumed. The venous and capillary pressures can rise greatly without approaching that of the larger arteries. However, if this compensatory force rises high enough to approach the level of the arterial pressure, a rise in the general arterial pressure will occur reflexly. Gradual or moderate increases in intracranial pressure may not interfere with the cerebral flow so long as the arterial pressure remains sufficiently high. To quote Loman: 'It is only when the brain is suddenly overwhelmed by great increases in intracranial pressure that the systemic arterial pressure responds to aid the flow of cerebral blood.'

4. *Intracranial Tension and the Classical Syndrome of Cerebral Compression.* The names of Kocher and Cushing are closely associated with the compression syndrome. The former, in his postulates, has set forth the sequence of events occurring as the result of increasing intracranial tension. He divided the course of compression into a series of stages:

Stage 1. A mild compression encroaches mainly on the venous bed, causing slight congestion and increase in capillary pressure.

Stage 2. Venous and capillary stasis are further increased, there is a relative anoxia of the vital bulbar centres, reflexly eliciting a slight rise in the systemic blood pressure, and a vagal slowing of the pulse rate. Early symptoms of compression become manifest: headache, excitement, stupor, possibly focal signs.

Stage 3. Advancing compression results in capillary anaemia. The medullary centres attempt to react by a marked increase in blood pressure and slowing of the pulse. Advanced symptoms now manifest themselves: respiratory changes, e.g. Cheyne-Stokes breathing, coma, pupillary and optic disc changes. The prognosis, previously good, now becomes grave.

Stage 4. Increasing pressure fails to elicit medullary reflex responses, i.e. the medullary centres become exhausted. The blood pressure falls, there is marked increase in the heart rate, the pupils become dilated and fixed, the respiration slows down

and finally ceases. This is the stage of decompensation and the prognosis is hopeless.

The above sequence of events has been accepted universally, though the fact that large numbers of cases of cerebral compression do not exhibit the classical syndrome demonstrates that it is unwise, even dangerous, to make the above teaching a dogma. When present, the signs are certainly important guides to the clinical assessment of the case, but their absence does not rule out a vicious expanding lesion. Experiments carried out by the writer have failed to confirm the classical march of events in compression.

Intracranial pressure can be increased either by subjecting the normal quantity of cerebrospinal fluid to increased tension (as by a space-occupying lesion); or by increasing the amount of fluid. The latter can be effected either by diminishing the rate of absorption, increasing the rate of secretion or by bleeding into the subarachnoid space. In addition, haemorrhage can interfere with the absorption of cerebrospinal fluid in several ways:

1. By mechanically blocking the arachnoid villi.

2. As a result of contusion and laceration of the brain there is a loss of absorption area for the pacchionian corpuscles (Shatara, 1936).

3. The communications between the intracerebral and extracerebral cerebrospinal fluid spaces are small and easily blocked and it is reasonable to assume, especially if coagulation occurs, that this may result in cutting off cerebrospinal fluid from the absorptive area.

Venous engorgement, a frequent accompaniment of brain swelling, results in increased intravenous pressure and interferes with normal fluid absorption, thus causing increased cerebrospinal fluid pressure. It is thought by some (Scott, 1940; Rand and Courville, 1931) that trauma may increase secretion due to abnormal activity or irritation of the choroid plexuses and ependyma.

Cushing showed experimentally that when the intracranial tension reached that of the arteries, the vasomotor centre responded reflexly, eliciting a rise in blood pressure, often to twice its normal level. Eyster (1901) proved that intracranial pressure of less than the mean blood pressure (90-100 mm. Hg) had very little effect upon blood pressure, pulse or respiration. Pressures encountered in trauma are rarely, if ever, capable of setting in motion the train of physiological events leading to medullary paralysis. Browder and Meyers (1936, 1938) find that a rapidly increasing intracranial pressure with its 'compensatory' syndrome has no application in clinical cases; and the reverse, viz. a compensatory syndrome, does not indicate increased cerebrospinal fluid pressure. In their experience repeated observations on the pressure cannot by themselves be employed as an index of the clinical condition of the patient, of his likely course, or of the necessity of surgical intervention. Such also must be the experience of most who are closely associated with this form of trauma. The conception of the classical compression syndrome so long taught can no longer be accepted in its entirety. Investigation of the course of severe and fatal head injuries have not upheld its tenets. There are indications that it is highly probable that many patients are losing their lives because their case sheets do not fall in line with the classical picture.

Zierold (1935) observed that when an abnormal intracranial pressure did occur, it was the result of haemorrhage, either extradural, subdural, subarachnoid or intracerebral. Crawford goes so far as to say that intracranial tension is not the basic problem and Russell (1932) found no evidence that increased pressure contributed to a fatal issue; low pressures are often found in the terminal stages of head injuries. Frequently successive daily punctures reveal pressures tending to normal when actually the patient was becoming comatose and operation demonstrated haematomata under quite considerable pressure (Brody, 1940). Incisural or foraminal blocks probably play important roles in these cases. Furthermore, a normal reading in a dehydrated patient (as these patients so often are) may be misleading, as the actual pressure is probably very much higher (Munro, 1938).

5. *The State of Consciousness and Intracranial Pressure.* One school of clinical observers thinks that unconsciousness *per se* is a most important gauge of the degree of intracranial pressure. Cannon, Scott, Gurdjian and Webster, to mention a few, experimentally or clinically proved high pressures to be related to unconsciousness. Denny-Brown and Russell (1941), however, found no evidence that increased pressure contributed to a fatal issue or that there was any relationship between the degree of unconsciousness and the spinal pressure. Zierold (1935) and Browder and Meyers (1938) likewise found no relationship between unconsciousness and pressure. Rowbotham (1949) found a moderately raised pressure in the majority of his unconscious patients. It is the writer's opinion, from an analysis of over 100 fatal cases, that the state of consciousness has no relationship to the degree of intracranial pressure as measured by lumbar manometry, *but this does not mean that there is no raised intracranial pressure or that some remediable space-occupying lesion is not present*—a fact that cannot be sufficiently emphasized.

Whether unconsciousness is due to disturbed thalamic function (Greenfield, 1938), frontal lobe function (Dandy), changes in the metabolism of neuronal cells (Walker, Kollross and Case), related to direct traumatic paralysis of the cortical neurones (Eden and Turner), anaemia of the brain (Trotter, Cushing, Scott), or injury to subcortical structures and the midbrain (Jefferson), there is no doubt of its profound bearing on the ultimate prognosis of the case. It is a most important indication when prolonged (12 hours or more), of the secondary effects of head trauma.

Loss of consciousness has always found its classical application in the development of the 'compression' syndrome. The lucid interval is the well-known feature of this development. The essential condition is the occurrence of a second phase of disturbance of consciousness, in which, owing to the gradual, or rapid formation of a space-occupying lesion, the patient lapses into the coma of compression, as distinguished from the unconsciousness of concussion. However, so frequently is the primary phase associated with severe disturbances of consciousness, that the secondary phase is a mere continuation, or superimposition, and no 'interval syndrome' is manifest. It is for this reason that any case showing prolonged unconsciousness must be regarded as due to a space-occupying lesion until proved otherwise by diagnostic craniotomy, and the diagnosis of the primary injury only verified by ruling out the secondary complication. The

second and third days constitute the critical period for unconscious patients (Hawkes, 1943). It is probably due to the fact that in this group the secondary manifestations are of rapid progress and contain the operable cases, in which failure to remove the cause of the compression, theoretically at any rate, inevitably leads to a fatal outcome. Munro goes so far as to state that no head injury should die after the third day!

6. *The Blood Pressure.* Browder and Meyers (1938) dispute the accepted dictum that increasing intracranial pressure produces its vital effects through stimulation of the medullary centres. Although experimentally the cerebrospinal fluid pressure must equal the systolic blood pressure before a rise in systemic blood pressure results, the clinical value of a change in blood pressure, according to these authors, was insignificant and possibly misleading. Dandy, Zierold, Eyster and Fremont-Smith and Merrit likewise found the blood pressure of little value in head injuries. Others, however (Denny-Brown and Russell, Walker *et al.*, Gurdjian and Webster, Pearce Baily) attach considerable importance to a rise in blood pressure, though Gurdjian (1933) himself found the pressure determination to be valueless as an index of increased intracranial pressure. It is probable, as stressed by Rowbotham (1949), that a rise in systolic pressure not infrequently occurs, often associated with a full, bounding pulse (due to the increase in pulse pressure), giving it the so-called 'vagal quality'. However, this rise is not always associated with a demonstrable rise in intracranial pressure and some other mechanism, in the nature of a vago-glossopharyngeal reflex, may play a part, possibly from the concussional injury.

7. *The Pulse.* The importance of the pulse rate has long been impressed on medical students and the so-called characteristic slowing has almost been accepted as a medical axiom—it is regarded as almost a travesty of medical supervision to omit the 'hourly pulse rate' chart. That occasionally a slowing of the pulse rate indicates exactly what it is meant to convey is not denied, yet not only can it be a false alarm, but the fact that this slowing has not occurred has been the reason for *not* suspecting increasing mischief of an operable nature and procrastination had inevitably led to verification on the post-mortem table or, too late, on the operating table. Of course, reliance on one sign alone in such a case is not good surgical practice, but it supersedes, in the mind of the practitioner, all the other signs of the 'compression syndrome', and it is rarely that one finds hourly recordings of blood pressure, temperature, respiration, states of consciousness, etc., all presumably showing characteristic variations as well. Kocher's postulates seems to have made of the pulse rate almost a mathematical formula, i.e. its variation inverse ratio to the intracranial tension. So many factors influence the circulatory system, however, that neither the pulse nor the blood pressure react to intracranial conditions with any degree of consistency (Vance, 1927). Marked bradycardia, apparently due to vagus excitation, can occur in simple concussion (Jefferson, 1938; Bagley, 1929). The occurrence, however, of a slow pulse in the stuporose or comatose patient is of important clinical significance: bradycardia, to be of any prognostic value, must always be considered in conjunction with the state of consciousness. Analysis of hourly pulse charts in a large series of fatal cases reveals practically always inconsistencies, the record showing a rate e.g. of 72, an hour later 64, then 80, 88, dropping down to the 60's and rising and falling irregularly until the terminal 'racing pulse' supervenes. Except for a few isolated instances, the pulse rate cannot be regarded as a reliable therapeutic index of intracranial traumatic pathology.

8. *The Respiration.* Classically, as in the case of the vasomotor centre, the respiratory centre responds to an increase in intracranial pressure (to the level of that in the arteries), manifesting itself in the well-known Cheyne-Stokes phenomenon. The respiratory centre, however, is more vulnerable and death in cranio-cerebral trauma is almost invariably ushered in by respiratory failure. The stertorous breathing seen in the severe head injury may be due to vagal inhibition or merely the generalized loss of tone found in all deeply unconscious patients. Typical Cheyne-Stokes respiration is unusual, but as the brain stem is a very vulnerable part in severe injuries, as manifested by the frequent occurrence of haemorrhages, it is not inconceivable that the *direct* effect of trauma on the respiratory centre either macroscopic, microscopic or neuronal, may play an important part in causing any type of respiratory variation and leading to a fatal issue.

9. *The Temperature.* Mild and moderate injuries are not characterized by any special disturbance in the temperature, which may rise to 100°-101° F. A rise in temperature has been one of the accepted findings in increasing intracranial pressure, similar to the other reactions of the vital centres, so much so that Stevenson, e.g. advocated 15-minute temperature readings, anything above 103° F being regarded as evidence of increasing intracranial damage from compressional effects. Blood in the cerebrospinal fluid may cause the temperature to rise to 102° F or even 103° F due to a 'sterile meningitis' or absorption of broken-down blood proteins; but when there is an early rise to this level or when it exceeds 103° F, it is indicative of serious interference with the thermo-regulatory centres.

Classically hyperthermia is attributed to haemorrhages in the pons (along with 'pin-point' pupils) and Courville (1945) considered focal haemorrhages, with or without oedema, in the midbrain or pons, to be frequent causative factors. By following up the clinical records of patients exhibiting these haemorrhages *post mortem*, the writer has not been able to verify these generally accepted dicta and there has been no correlation between pontine haemorrhages and the alleged hyperpyrexia.

Vomiting. Occurring early, this is usually due to recovery from the concussive phase, to the gastric irritation of alcohol or, in some cases, from ingested blood. In the later phase of cranio-cerebral trauma it may be significant, yet most authorities (e.g. Zierold; Browder and Meyers) disregard its importance as evidence of raised intracranial pressure. Vomiting in the deeply unconscious patient, however, is gravely ominous, often enough preceding death.

10. *The Reflexes.* Two series of reflex changes occur:

1. Those related to the actual concussive phase;

2. Those associated with the development of secondary complications.

In regard to the former, loss of the corneal reflex is

significant of a sensori-motor disturbance of the medulla, occurring at the time of the acute traumatic disturbance of cerebral function. During the deep coma of primary injury the limbs are generally completely flaccid and all the superficial reflexes are usually abolished. A characteristic feature, however, is the variability of the reflexes, including the plantar reflex, so much so that they have been regarded as being of no significance (MacGregor, 1942).

When complications, particularly haemorrhage, become superimposed, clinically very valuable alterations may occur. The manifestation of unilateral hyper-reflexia is very often indicative of the development of local compression, especially when associated with significant *pupillary changes*. In this respect it is the unilateral and, in the late case, bilateral fixed dilatation of the pupils that is the most important change. The mechanism is probably as follows: As the uncus descends in a supratentorial herniation, it compresses the third nerve or stretches it (with the slight downward displacement of the midbrain). The effect on this nerve, containing the parasympathetic fibres of the sphincter mechanism of the pupil, is at first to stimulate it, causing a slight transient phase of pupillary constriction. Increasing pressure paralyses the sensitive parasympathetic fibres, leading to sympathetic overaction and a gradual dilatation of the pupil. During this phase there may be a sluggish reaction to light. Finally the pupil becomes fully dilated and fixed. By this time the brain stem is being compressed against the opposite edge of the incisura tentorii (or the opposite uncus commences to herniate), giving rise to a similar chain of effects until finally both pupils are widely dilated and completely immobile. Intrinsic primary or secondary changes in the brain stem may play a part in disturbing the oculomotor mechanism. In some late cases, complete third nerve paralysis may occur, resulting in partial ophthalmoplegia and ptosis. An expanding lesion in the spheno-temporal region is particularly dangerous in initiating uncal herniation, usually on the same side, and thus causing an ipsilateral fixed dilated pupil, indicating the site for initial exploration. It is not unusual to find spasticity and hyper-reflexia on the same side as the dilated pupil. In these circumstances it is postulated that the brain stem is compressed against the opposite sharp edge of the incisura, causing a lesion of the opposite pyramidal tracts and thus ipsilateral reflex and other changes; hence the advice to explore first on the side of the dilated pupil.

11. *Anoxia and its Effects on the Brain.* The brain is extremely avaricious for O_2, particularly the grey matter, which utilizes four times as much as the white (accounting for the greatly increased capillary bed in the former). It can only use up energy derived from carbohydrates, principally in the form of glucose, this substance and O_2 being taken up in approximately equal amounts. By phosphorylization glucose is transformed, in a series of steps, ultimately to pyruvic acid, and this again, in an adequate supply of O_2, to CO_2 and water. If there is insufficient O_2, the process is incomplete and pyruvic acid is converted to lactic acid. Cerebral anoxia has been shown to cause an increase in the cerebral lactic acid; likewise thiamine deficiency and, to a certain extent, most anaesthetics. There is now sufficient evidence to show that anoxia plays an integral part in the physio-pathology of cranio-cerebral trauma and may be an important contributory factor to a fatal outcome. Cerebral trauma *per se* affects the circulation to and from the cells, besides which shock, oedema, anaemia, hyperthermia and drugs all tend to one or other form of anoxia, further increasing the damage to the brain, the cells of which are extremely vulnerable to O_2 deprivation. Any of the different types of anoxia may affect the tissues of the brain. Thus anoxic anoxia results from congestion, oedema or consolidation in the lungs. Anaemic anoxia occurs where the head injury is accompanied by severe haemorrhage (as from scalp wounds) or in an atmosphere of carbon monoxide (as in mine accidents). Stagnant anoxia is present in conditions of shock but more particularly as a result of the congestion and stasis so frequently associated with severe head injuries. Histotoxic anoxia, in which there is a direct interference with tissue utilization of O_2, could be particularly evoked by the undue administration of narcotics or anaesthetics. In the severely traumatized patient the combination of all the above factors plays no small part in the fatal outcome of the case. Depression of the blood O_2 can range from 5% to as much as 44% (Schnedorf *et al.*, 1940). The most permeable section of the small vessels appears to be at the point of transition between capillaries and venules. In stasis (possibly consequent to vasoparalysis—Scheinker, 1944) anoxic damage to the vessel wall increases its permeability, allowing the escape of the fluid and formed elements of the blood into the perivascular spaces, thus further compressing the vessels and causing a vicious circle of congestion and stasis. This is enhanced by the accumulation of abnormal metabolites in anoxic tissue causing the latter to absorb more fluid and accentuating the already existing oedema (Masserman, 1934).

(To be continued)

INFANTILE ECZEMA*

S. GORDON, M.B., CH.B. (WITS.), M.R.C.P. (ED.)
Johannesburg

The subject of infantile eczema should be approached with humility. There is so much about it that is not known and so much that passes as knowledge which is no more than the cumulative repetition of a hundred textbooks.

The word eczema has come down to us from antiquity and has unfortunately brought its antiquity with it, so much so that the whole subject is still encrusted with the barnacles of pseudo-medical terminology.

Dermatologists have laboured for many years to raise the status of eczema to the level of an ordinary medical phenomenon—often at cross purposes, often confusing

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elaboration with clarification, and always a little aghast at their own temerity. All these labours have at times added a little to our knowledge, sometimes they only added to our understanding, and often they only served to bring our ignorance into sharper relief. On the basis of the little knowledge that we now have and with the aid of some legitimate inference, it is to-day possible to adopt, in outline, a classification of eczema to which future investigation will give substance and shape.

If we accept some such classification—and we must, if we are going to particularize about infantile eczema—we begin with a definition of eczema as an inflammatory reaction of the epidermis to any substance to which it has become sensitized. By the terms of this definition the choice of an eczema subject is not necessarily determined by his age, heredity, diathesis or constitution. Similarly the sensitizing substance is not exclusively an external contactant, or an ingested substance of known composition, or an endogenous toxin of unknown nature. All that is necessary for an eczema reaction to take place is that the eczema subject and the sensitizing substance should have met and developed a mutual antagonism. Henceforth, whenever they meet, they will recognize each other and display that antagonism in the form which has become familiar to us as an eczema reaction.

Based broadly on this definition is a classification of the eczemas into the following groups:—

1. *Exogenous Eczema.* Here the reaction depends on a known and demonstrable contactant.
2. *Infective Eczema.* In this the sensitizing substance is bacterial or fungal and produces the reaction also by external contact.
3. *Endogenous Eczema.* In this the sensitizer is either a known substance which has been ingested, injected or inhaled or, by inference, a circulating toxin of unknown nature and doubtful existence.

This classification meets with difficulties when it comes to the atopic eczemas, which are said to be more common in some families than in others and in which there is a multiple sensitivity which may be both endogenous and exogenous.

To a very large extent the clinical picture of an eczema shows a corresponding morphological division. Thus an exogenous chemical eczema is commonly diffuse and vesicular, an infective eczema is usually a sheet which increases by peripheral extension, and an endogenous eczema is discoid and papular; but this is no more than a rough differentiation and should not be regarded as a complete guide to etiology. The essential unity of the eczema reaction is best demonstrated by the sulpha drugs which can produce the same eczematous rash when taken by mouth or applied topically. It is also illustrated by the behaviour of certain external irritants, notably nail polish, which can produce a diffuse eruption in some individuals and discoid scaly patches in others.

Such a classification of eczema has at last hesitantly insinuated itself into the pages of conservative dermatology,[1] but the benefit of rational classification has not yet been accorded to infantile eczema. This is still heavy-handedly described as a single entity, nebulously attributed to endogenous toxins, allergens or atopens. This is over-simplification at its worst. To accept such a single etiology is to deny that an infant is subject to the same laws of antigen-antibody reaction as an adult. Percival,[2] who goes a little further than other writers, admits the existence of other forms of eczema in infants and distinguishes between eczema in infants and infantile eczema. This is an unnecessary complexity. Once it is admitted that exogenous forms of eczema do occur in infants, then for all practical purposes every case of infantile eczema becomes an eczema in an infant and may belong to any of the groups that we recognize in adults.

Following the same sequence that we adopted for adult eczema, infantile eczema can now be discussed under the following headings:

Contact Eczema. As a baby does not use cosmetics and is not exposed to industrial hazards it escapes the two common sources of exogenous sensitization and the incidence of contact eczema in infants is almost negligible; almost, but not quite. F. A. Simon [3] described a contact sensitivity to the mother's dandruff which could be demonstrated by patch tests. Gaul and Underwood [4] described a generalized eczema reaction in a baby as a sequel to sensitization by footwear. A personal case is that of a baby sensitive to the mother's hand lotion. The importance of recognizing this group, however, lies in the fact that infants are daily exposed to the risk of sensitization by skilfully advertised ointments of questionable value and high sensitizing potential. As long as mothers will rub patent ointments into flea bites and pour drops into stuffy noses, the possibility of exogenous sensitization will always be with us. Moreover, the use of such ointments is liable to convert a mild bacterial or endogenous eczema into a severe chemical eczema.

Infective Eczema. It has been recognized for many years that some infantile eczemas start with a cradle cap, show an early tendency to affect the retro-auricular area, the axillae, and the groin, and appear as sharply marginated, bright red, oozing and crusted sheets. Misguided attempts have been made to bring these within the orbit of seborrhoeic eczema. As seborrhoeic eczema has only a mythical existence in the adult, and as infants do not suffer from greasy skins, or seborrhoea, this term can hardly be regarded as aptly descriptive. The recognition of this form of eczema as a bacterial infection and bacterial sensitization is a real advance in dermatology and the term seborrhoeic eczema, in general, should now be allowed to go to its final resting place and forgotten. Unfortunately, the victims of sulphosalicylic ointment therapy will have reason to remember it for some time to come.

There is a definite relationship between impetigo and eczema in infants. This is important because all forms of impetigo are common in infants. There is the acute impetigo contagiosa; there is the chronic streptococcal impetigo, which starts from a discharging ear; there is the fine scaly impetigo of the face and the more generalized scaly impetigo of the trunk which has been described as erythema streptogenes.[5] Another form of impetigo, which appears as circumscribed, finely crusted, erythematous patches and is often mistaken for eczema was described by Adamson [6] in 1908 and more recently by Crawford [7] as chronic symmetrical impetigo. These forms of impetigo are mentioned here not only because they can be mistaken for eczema, but because in certain circumstances they become complicated by an eczema reaction. This brings us to the subject of auto-eczematization.

Under the term autosensitization Whitfield [8] described

the appearance of widely distributed papulo-vesicular patches a number of days after an exacerbation of a chronic localized, lichenified eczema. Recently Cormia and Esplin [9] described a similar phenomenon which they call auto-eczematization. Briefly their concept is that epidermal cells in a state of inflammation may become antigenic and produce widespread epidermal reactions. This process is enhanced by chemical irritation or mechanical trauma. Cormia and Esplin believe that liberation of antigens may be determined by bacterial infection and chemical sensitization of a limited area of skin. They suggest that clinically distinct entities, e.g. nummular eczema, atopic eczema and infective eczema may have this mechanism in common. In fact, they hope that this concept will unravel the enigma of eczema.

The application of this concept to infantile eczema is very obvious. Its most practical application is in this group of infective eczemas. It is only necessary to accept the hypothesis that epidermal cells altered by bacterial infection may acquire antigenic properties to have an explanation for the development of eczema as a sequel to an impetiginous infection of the skin. Add to this the dangers of auto-eczematization by chemical irritation and the trauma of scratching and one begins to wonder whether this sequence does not account for a large part of infantile eczema. Personally, I think it does.

Atopic Eczema. Under this heading may be conveniently grouped all eczema reactions, without discoverable exogenous causes. Here we come to the submerged part of medicine where a little speculation is permissible and is, indeed, inevitable. For the sake of simplicity atopy may be defined as an inherently low sensitivity threshold. By this definition persons belonging to certain families are liable to acquire a polyvalent sensitivity to a variety of sensitizers, or atopens, the effect of which is the development, among other sensitivity phenomena, of an eczema which appears early in life and may persist throughout childhood and into adult life. Clinically this form of eczema is a dry, papular, often lichenified eruption in somewhat ill-defined sheets affecting the face, the antecubital and popliteal areas, the ankles and the wrists. Etiologically this group is very ill-defined. It cannot even be regarded as purely endogenous, because sensitivity to contact with woollen garments is known to be a factor. However, to many dermatologists and pediatricians atopic eczema is almost synonymous with food allergy and calls for an immediate assault on the baby's diet. In terms of our definition of eczema it must be accepted that some eczema reactions are caused by food allergens, but this over-emphasis of food allergy would lose much of its background if the subject were reviewed and all the evidence based on skin allergy tests were finally and unhesitatingly discarded.

There are some perplexing questions in connection with food allergy to which the answer is not obvious. Firstly, if food allergy is a common cause of eczema in infants, why is it that urticaria, the more familiar manifestation of food allergy, is so uncommon in infants? Secondly, food allergy as a cause of eczema is now well recognized in adults. Why is it then that all the investigators in this field[10] have found that the maximum incidence is as an eruption of the hands and not of the areas commonly affected in atopic eczema? Thirdly, what is that phenomenon which takes place at the age of two or three and which produces a sudden and permanent disappearance of the sensitivity in the majority of infantile eczemas? In the present state of our knowledge there is not much more that can be said about atopic eczema, except to refer again to an observation that has been made before, that atopic eczema has many points of similarity with dermatitis herpetiformis.[11] Dermatitis herpetiformis was described by Duhring in the last century [12] and since then it has gradually come to mean an itchy, pigmented, bullous eruption, but this imposes a limit on Duhring's work which Duhring never intended it to have. Duhring also described other types of dermatitis herpetiformis, including a papular eczematous type. Between this papular type and the papular type of eczema the dividing line becomes so blurred that it is a reasonable speculation that dermatitis herpetiformis, atopic eczema and auto-eczematization are either the same or closely related phenomena. The importance of this will be seen when we come to treatment, but before we do that it might be convenient at this stage to mention two other conditions which hover on the horizon to catch the unwary. One is prickly heat. This is an itchy eruption of fine translucent papules on the covered parts of the body. The affected skin feels as if it had been dusted with fine sand. It is dealt with very simply by stopping the baby's bath for a week or so and using a mild salicylic lotion. The other is a non-itchy fine papular eruption which often follows an upper respiratory infection. It is regarded as a streptococcide and responds to sulphonamide therapy.

The treatment of infantile eczema follows on naturally from the classification which has been adopted. In the contact-type exogenous eczema, all that is necessary is to eliminate the offending allergen and apply a bland lotion. It must be emphasized that general sensitization is always imminent and that the thoughtless application of 'panacea' ointments to this type of eczema almost amounts to malpraxis. The infective type of eczema is dealt with primarily by the topical application of antiseptics of low sensitizing potential. The applications that deserve special mention are Tyrothricin solution 0.1% which is used as a wet dressing, and Vioform in 3% concentration as a lotion, cream or paste, depending on the stage of the eczema reaction. While not a great adherent of polychromatic dermatology, I must admit that some of the dyes, e.g. ½% brilliant green, have their uses in this group. After a period of antiseptic treatment, when the eruption has largely subsided and the skin has become dry and scaly, it is often useful to apply a crude coal tar paste. Incidentally, applications of crude coal tar paste have a not undeserved reputation in all forms of eczema, when this particular stage has been reached.

In atopic eczema the external treatment is only of secondary importance. As in the infective type, the application of some antiseptic in the presence of secondary impetigo is helpful and the application of crude coal tar may relieve the itching, but this does not influence the atopic state.

A new approach to the treatment of the atopic state has emerged as a result of the observation of the similarity between atopic eczema and dermatitis herpetiformis. The

etiology of dermatitis herpetiformis is as much a mystery as the etiology of eczema. What is known about dermatitis herpetiformis is that the eruption can be kept suppressed indefinitely by arsenic, or Sulphapyridine, or para-aminobenzoic acid. It is also known that suppression of the condition for long periods will in many cases bring about an eventual cure.[1]

After an extensive trial in cases of atopic eczema and auto-eczematization it can now be added that the similarity between dermatitis herpetiformis and atopic eczema is even more striking in the field of therapeutics. Without trying to explain the rationale of this treatment, without claiming infallibility for this method, it can now be stated that the administration of Sulphapyridine or Pabavel has proved of inestimable value in the management of atopic eczema in infancy. Until more is known about the anti-eczematous properties of these substances, it is only possible to say that their action on the epidermis is comparable with the action of antihistaminic drugs on the dermis.

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IN MEMORIAM

DR. J. S. MORTON

Dr. James Stevenson Morton, the doyen of the medical profession on the East Rand, died on Saturday, 21 July 1951, at the age of 84.

He was born in Northern Ireland and qualified at Trinity College, Dublin, obtaining the M.B. Ch.B. and subsequently proceeded M.D. He came to South Africa in 1894 as Medical Officer to the Farrar Group of Mines, a position he held jointly with the late Dr. J. McNeillie. During the Boer War he took part in the siege of Kimberley. At the end of this war he returned to Boksburg, becoming Medical Officer to the E.R.P.M. Ltd. He was also employed as Medical Officer to Brakpan Mines from 1906 to 1908 and the Geldenhuis Deep for a short period.

For many years he held the post of Senior Honorary Physician to the Boksburg Hospital and subsequently to the Boksburg-Benoni Hospital, retiring from this position in 1936. He was the Medical Officer of Health to the Boksburg Municipality for a number of years. For many years he was a valued member of the Boksburg-Benoni Hospital Board.

He was a keen member of the B.M.A. and numbered amongst his friends many of the leading surgeons of Johannesburg. In 1944 he was made an Honorary Member of the B.M.A. after 50 years of unbroken membership and subsequently he was similarly honoured by the local East Rand Division and East Rand Branch of the M.A.S.A. On the latter occasion he delivered a stimulating address on 50 years of practice on the East Rand, showing a keen appreciation of modern developments and trends in medicine.

He held high office in the Masonic world; was a keen and able golfer and his company was much sought after on the golf course. In his later years he became a keen bowler and proved himself adept at this game.

He continued in practice up to his last illness. A man of kindly disposition, he was esteemed by patients and colleagues alike.

G. B. M.

* * * *

A correspondent writes: The death occurred in Boksburg, Transvaal, on 21 July 1951, of James Stevenson Morton, M.D., D.P.H., aged 84 years. He was the elder son of Mr. James Morton, of Mount Pleasant, Gilford, Northern Ireland, and was born on 18 April 1867. He was educated at the Belfast Academy and then at Trinity College, Dublin, where he obtained his Medical degree. He proceeded to 'The Rotunda' for midwifery, and later took his D.P.H. at Cambridge.

Dr. Morton came out to South Africa in 1895, arriving in Boksburg on 24 December of that year. He was granted a licence to practise Medicine in the Transvaal Republic under President Kruger. He joined the British Forces in 1899 at the outbreak of the Boer War, and was in Ladysmith throughout the historic siege. After the Relief, he was appointed to a Hospital Ship plying between South Africa and Britain where he served until the Armistice in 1902, when he returned to Boksburg.

He was appointed M.O.H. for Boksburg in 1905 and served in that capacity until 1936. He did honorary work for the Boksburg-Benoni Hospital from the time of its inception until very recently, and apart from his general practice, was one of the E.R.P.M. (East-Rand Proprietary Mines) doctors.

Dr. Morton was a prominent member of the Masonic Lodge in Boksburg and took a keen interest in all public affairs. He continued in general practice until his last illness, in June this year, having been practising in Boksburg for 56 years. He leaves a widow and one daughter, his only son having been killed in action in the Royal Navy in 1945.

PASSING EVENTS : IN DIE VERBYGAAN

We regret to record the death of Dr. M. A. Diemont, of Stellenbosch, who passed away on 1 September 1951.

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We regret to record the death of Dr. J. A. Weir, a general practitioner of Rondebosch, Cape, who passed away on 4 September 1951.

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Dr. Percy Helman of Cape Town has been successful in the Primary Examination for the Fellowship of the Royal College of Surgeons, England.

TENTH INTERNATIONAL CONGRESS OF DERMATOLOGY : 1952

The Academic Committee is now prepared to receive applications from dermatologists who wish to read short papers at this Congress. Speakers will have 15 minutes in which to present their papers. In the majority of instances this will be followed by a discussion not exceeding 15 minutes.

Chairmen of Meetings will be asked to insist on the 15-minute schedule.

Titles of communications and an accurate note concerning the name, appointments and degrees of the applicant should be forwarded to the Congress through Dr. J. Frootko, 1006 Medical Centre, Jeppe Street, Johannesburg, by 1 January 1952.

* * * *

Dr. C. C. Pretorius is terug van sy nagraadse studies oorsee, en spesialiseer nou as verlos- en vrouesiektekundige te Transvaliagebou 416, Sentraalstraat, Pretoria.

Provincial Administration of the Cape of Good Hope

HOSPITALS DEPARTMENT

HONORARY APPOINTMENT

Applications are invited from registered medical practitioners for the following post:

Honorary Surgical Registrar at the Victoria Hospital, Wynberg.

The appointment will be for the remainder of 1951.

Applications containing particulars of age, qualifications, experience, etc., with copies of recent testimonials should be forwarded to the undersigned by Noon, 29 September 1951.

L. Welham
Branch Representative

Hospitals Department
Industry Building
58 Loop Street
Cape Town 4734

Transvaalse Provinsiale Administrasie

VAKATURES BY PUBLIEKE HOSPITALE

Aansoeke word ingewag van kandidate met geskikte kwalifikasies vir die onderstaande pos by die Ontdekkersgedenkhospitaal, P.K. Florida in die Transvaal.

Aansoeke met gerig word aan die Verantwoordelike Geneesheer van die Ontdekkersgedenkhospitaal en moet volle besonderhede bevat aangaande die ouderdom, professionele, akademiese en taalkwalifikasies, ondervinding en huwelikstaat van die applikant en moet voorts 'n aanduiding bevat van die vroegste datum waarop diens aanvaar kan word.

Ontdekkersgedenk:—Deeltydse Algemene Praktisyn/Chirurg, £225 p.j. moet hospitaal daagliks besoek.

Van die persone wat aangestel word, sal verwag word om bevredigende sertifikate in te dien.

Aansoekvorms is verkrygbaar van die Provinsiale Sekretaris, Departement van Hospitaaldienste, Posbus 383, Pretoria.

Die sluitingsdatum van aansoeke vir die pos is 2 Oktober 1951. (30923)

Royal College of Surgeons of England

PROFESSORSHIP OF HUMAN AND COMPARATIVE ANATOMY

The Council invites applications for the Sir William Collins Professorship of Human and Comparative Anatomy.

The salary will be from £2,000 to £2,500 per annum according to age and academic status with arrangements for membership of the Federated Superannuation System for Universities.

The election is an open one, but the Council reserves the right to invite individual applications.

Particulars and terms of appointment may be obtained from the Secretary. Applications with the names of two referees must reach the College not later than 30 November 1951.

Kennedy Cassels
Secretary
Lincoln's Inn Fields
London, W.C. 2, England 4 RC/C 58

Vakature vir Mediese Beampte

Die aandag word gevestig op 'n advertensie in die *Staatskoerant* van 14 September 1951, waarby aansoeke om aanstelling gevra word, op kontrak, in 'n vakante pos van Mediese Beampte in die personeel van die Gesondheidsinstituut vir Gesin en Samelewing, Clairwood, Durban.

Die salarisskaal aan die pos verbonde, is £720 - 30—900 - 40—£1,020, plus 'n lewenskostetoelae van £256 per jaar in die geval van getroude en £80 per jaar in die geval van ongetroude amptenare.

Aansoekvorms is verkrygbaar van die Sekretaris van Gesondheid, Posbus 386, Pretoria. (30872)

The Medical Association of South Africa
Die Mediese Vereniging van Suid-Afrika

AGENCY DEPARTMENT : AGENTSKAP AFDELING

KAAPSTAD : CAPE TOWN

Posbus 643, Telefoon 2-6177: P.O. Box 643, Telephone 2-6177

PRAKTYKE TE KOOP : PRACTICES FOR SALE

(644) Durban Central. Mainly Indian and Native cash practice. Average annual gross income £1,235. Premium of £500 required for goodwill, inclusive of furniture and fittings and drugs. Terms may be arranged.

(805) Transkei Native and D.S. practice. Near large town. House and surgery for sale, purchaser preferably bilingual.

ASSISTENTE PLAASVERVANGERS VERLANG ASSISTANTS LOCUMS REQUIRED

(754) For Transkei Native and D.S. practice. Initial salary £60 p.m. and all found. Definite view to partnership after trial period. Single man preferred.

(724) Cape Town. Excellent opportunity for assistant in Northern suburbs. Salary, etc., to be arranged.

(744) Noordweste. Assistent vir praktyk met klein private verpleeginrigting. £65 p.m. plus vry losies. Kar beskikbaar.

(792) Cape Town Southern suburb. Middle- and better-class European practice. From 1 December for 4½ weeks. £2 2s. p.d. plus 10s. car allowance p.d. plus board and lodging. Must have done some general practice previously. Very little night work.

(723) Assistantship in hospital town near Cape Town. Salary £75 p.m. and car provided. Dwelling available. Possibility of permanent appointment.

(798) Cape Town. From 14 October for three weeks. £3 3s. per day. Locum to use his own car and live at principal's home.

(891) South-Eastern Cape. Large partnership in hospital town requires a locum as from 1 October. Preferably Afrikaans and married. £2 2s. per day plus all found, allowance for use of car. Possibility of long-term appointment.

* * * *

JOHANNESBURG

Medical House, 5 Esselen Street. Telephone 44-9134-5, 44-0817
Mediese Huis, Esselenstraat 5. Telefone 44-9134-5, 44-0817

PRAKTYKE TE KOOP : PRACTICES FOR SALE

(Pr S30) Johannesburg Partnership practice plus Solus practice. Mainly non-European. Present income £3,600 p.a. Premium for quick sale £1,250.

(Pr S31) O.V.S.-praktyk. Goeie geleentheid vir algemene geneesheer met aanleg vir snywerk. Alle fasiliteite. Medisyne word aangemaak. Moet tweetalig wees. Jaarliks inkomste £2,400. Eienaar gaan verder studeer. Premie vir klandiesiewaarde, instrumente en voorrade, £1,500. Een maand introduksie sal gegee word.

ASSISTENTSKAP VERLANG : ASSISTANTSHIP REQUIRED

(A W52) Assistantship in established practice in S. Rhodesia required by married South African doctor. Qualified two years.

ASSISTENT/PLAASVERVANGER VERLANG ASSISTANT/LOCUM REQUIRED

(L V112) Johannesburg from 18 December 1951 to 18 January 1952. £2 12s. 6d. p.d. plus all found and petrol and oil paid.

MEDICAL EQUIPMENT

(I 017) Leitz microscope £45.

(I 018) What offers? Complete set 'British Encyclopaedia of Medical Practice' plus all editions of 'Medical Progress' to 1950. Condition as new.

(I 019) Zeiss microscope. Condition as new. £55.

(I 020) "Standby" model Baumanometer. £10.

(I 021) Portable Baumanometer. £3.

(I 022) Klinostik Auroscope with Ophthalmoscopic attachment. £3.

(I 023) Heavy based Irrigator stand, height adjustable, complete with glass flask and hook to carry vacolitre flasks. £7.

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